BILATERAL PUTAMINAL HEMORRHAGIC NECROSIS FOLLOWING METHANOL POISONING

Radu Mihailescu[1], Elena Copaciu[2], Corina Pop[3], Amalia Ene[4], Bogdan Ovidiu Popescu[1,5]

[1]Department of Neurology, Colentina Clinical Hospital, CDPC, Medical School, „Carol Davila" University of Medicine and Pharmacy, Bucharest
[2]Department of Intensive Care, University Emergency Hospital, Bucharest
[3]Department of Internal Medicine, University Emergency Hospital, Bucharest
[4]Department of Neurology, University Emergency Hospital, Bucharest
[5]Laboratory of Molecular Medicine and Neuroscience, "Victor Babes" National Institute of Pathology, Bucharest

INTRODUCTION

Although it shares a resemblance with ethanol in both looks and organoleptic properties, methanol is a highly dangerous alcohol. Intoxication with it can lead to severe metabolic acidosis, blindness, neurologic impairment and possiblydeath. Even though generally rare, putaminal necrosis, with or without hemorrhage, is the most common radiologic finding.

CASE REPORT

A 60 year-old male, with a history of chronic kidney disease, diabetes mellitus and alcohol abuse, presented with abdominal pain, vomiting and slight confusion, symptoms soon culminating in a respiratory arrest. He was admitted in the Intensive Care Unit with severe wide anion gap metabolic acidosis, hyperglycemia, high lipase levels and a Glasgow Coma score of 4 (E1V1M2). The neurologic examination, required the following day for the persistence of the comatose state, revealed a GCS=6 (E1V1M4), non-reactive bilateral miosis and a bilateral Babinski sign; the foveal reflex was absent at fundoscopy. The brain CT scan showed bilateral putaminal hemorrhagic necrosis. The patient´s family later confirmed the suspicion of methanol intoxication (initially overlooked because of the presumed diabetic etiology of the acidosis), admitting the consumption of windshield washer fluid. Following a slowly favorable evolution, the patient was discharged after two weeks with complete blindness and mild global bradykinesia.

CONCLUSIONS

The classic radiological finding described in methanol intoxication is bilateral putaminal necrosis. Many theories have tried to explain this selectivity, among which a higher metabolic demand of the basal ganglia, a predisposition to watershed infarcts due to their lying in arterial junction territories, a deficient venous drainage leading to a higher concentration of toxic metabolites (1,2,3,4) or a higher concentration of formic acid (the highly toxic end product of methanol metabolism) or an increased vulnerability to it or to acidosis in the putaminal neurons (5). It was also found that methanol elevates cytosolic calcium ions in cultured canine cerebral vascular muscle cells, which suggest that cerebral vasospasm may be induced by methanol through a rise in intracellular calcium (6).

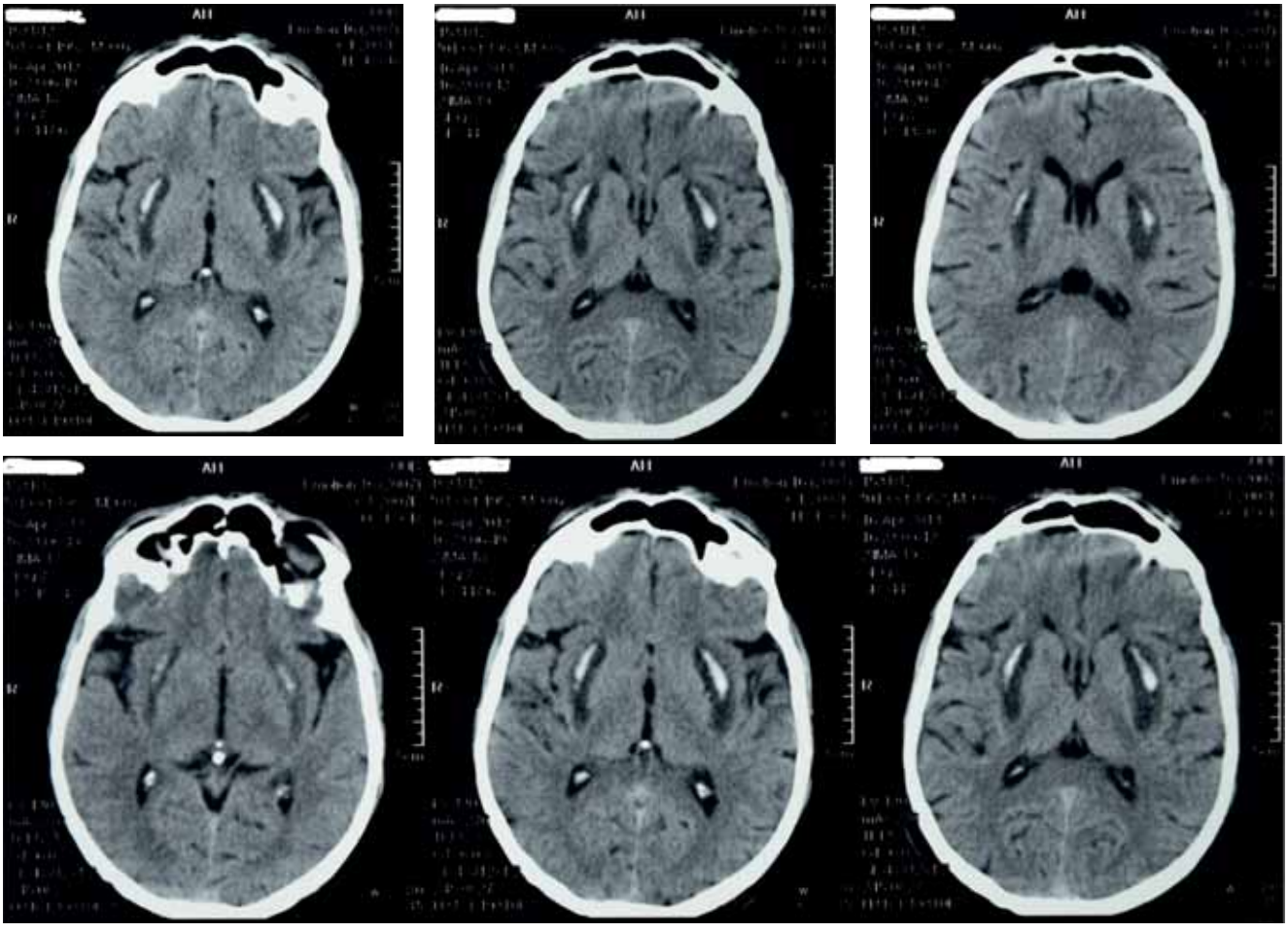
The hypoxic episode may have as well contributed in our case to these lesions. In adults, putaminal necrosis may also be seen in hypoxic-ischemic

Author for correspondence:
Radu Mihailescu, MD, 2nd Neurology Department, Colentina Clinical Hospital, 19-21 Stefan cel Mare Str., Bucharest
E-mail: radanmih@yahoo.com

injury and trichloroethane, ethylene glycol and carbon monoxide poisoning. However, in carbon monoxide poisoning, thetoxicity usually targets the globus pallidus, while hypoxic injuries additionally involve the caudate nucleus and other central gray nuclei (7).

While methanol poisoning should always be borne in mind as a cause of high anion gap metabolic acidosis, it may be overlooked when diseases causing such blood gas changes (like diabetes mellitus) coexist.

Methanol intoxication must always be considered in patients with metabolic acidosis and neurologic deficits. It should be taken into account as a rare cause of parkinsonism as well.A prospective study should determine the role of routine brain imaging in all severe methanol poisoning cases.



REFERENCES

1. **Hsu H.H., Chen C.Y., Chen F.H., et al.** Optic atrophy and cerebral infarcts caused by methanol intoxication: MRI. *Neuroradiology* 1997; 39:192-94
2. **Gaul H.P., Wallace C.J., Auer R.N., et al.** MR findings in methanol intoxication. *AJNR Am J Neuroradiol* 1995; 16:1783-86
3. **McLean D.R., Jacobs H., Mielke B.W.** Methanol poisoning: A clinical and pathological study. *Ann Neurol* 1980; 8:161-7
4. **Sharpe J.A., Hostovski M., Bilbao J.M., Rewcastle N.B.** Methanol optic neuropathy: a histopatologic study. *Neurology* 1982; 32:393-408
5. **Liesivuori J., Savolainen H.** Methanol and formic acid toxicity: Biochemical mechanism (review) *Pharmacol Toxicol.* 1991; 69:157-63
6. **Li W., Zheng T., Wang J., Altura B.T., Altura B.M.** Methanol elevates cytosolic calcium ions in cultured canine cerebral vascular smooth muscle cells: possible relation to CNS toxicity. *Alcohol* 1999; 18(203): 221-4 (abstract)
7. **Koopmans R.A., Li D.K., Paty D.W.** Basal ganglia lesions in methanol poisoning: MR appearance. *J Comput Assist Tomogr* 1988; 12:168-9